I realize the above is not earthshaking news, but it is submitted with the hope that medical writing will continue to use words that have some meaningful relation to the rest of the language.

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REFERENCE

1. Mathews WC, Booth MW, Turner JD, et al: Physicians' attitudes toward homosexuality—Survey of a California county medical society (Information). West J Med 1986 Jan; 144:106-110

EDITOR'S NOTE

Another dictionary *(Webster's Ninth New Collegiate)* defines homophobia and homophobic as "irrational fear of homosexuality or homosexuals." However, Mailman's point is well taken. The headings in the table might have been changed to "Positive, Neutral, Negative."

MSMW

An In Vivo Assessment of the Brain in Alzheimer's Disease

TO THE EDITOR: Alzheimer's disease is a neurodegenerative disorder accompanied by loss of neurons and their connections in selected brain areas. The loss of interneuronal structural integrity represents a fundamental defect that prohibits the normal communication between nerve cells. It is this communication that is the essence of nervous system function.

Current evidence now implicates the acetylcholine neurotransmitter system as one of the principal areas of damage in Alzheimer's disease. A number of features further characterize the several aberrations germane to the neuropathology of Alzheimer's disease. Some of these are a decrease in choline acetyltransferase found in the cortex upon postmortem examination[1] and a reduction in the apparent synthesis of acetylcholine in biopsy tissue[2]; also, the basal forebrain nucleus of Meynert,[2] which sends cholinergic circuits to widespread areas of the cerebral cortex, has been shown to display remarkable neuronal losses in the brains of patients with Alzheimer's disease.

Despite these important developments in our understanding of the neuropathology in Alzheimer's disease, we still remain unable to specifically diagnose the disorder clinically. Most current diagnostics fall under the rubric of the medical exclusion criteria with its groundings based upon clinical examination and psychometric testing. Furthermore, under the best of circumstances, which are difficult to achieve, diagnostic accuracy does not exceed 90%.

Recently a group in Boston and Washington, DC,[3] has reported the first utilization of in vivo imaging of receptor binding and cerebral perfusion in a patient with Alzheimer's disease. Using a technique called single photon emission computed tomography (SPECT) and iodine 123-labeled 3-quinuclidinyl-4-iodobenzilate (123-QNB) and perfusion imaging by employing ^{123}I-*N*-isopropyl *p*-iodoamphetamine, these groups have demonstrated that a patient with Alzheimer's disease exhibited a sharp decrease in perfusion to the posterior temporal and parietal cortex and a moderate diffuse impairment of muscarinic receptor binding, which cannot be attributed to perfusion changes.[4] Until now, receptor function could be evaluated only at biopsy or autopsy. The development of radiotracer techniques such as specifically labeled agonists and antagonists of particular neurotransmitters in concert with emission tomography can potentially provide clinicians with an invaluable tool to explore for the in vivo assessment of receptor binding and cerebral perfusion in clinical investigations.

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REFERENCES

1. Davis P: Neurotransmitter related enzymes in senile dementia of Alzheimer type. Brain Res 1979; 171:319-327

2. Bartus T, Dean RL, Beer B, et al: The cholinergic hypothesis of geriatric memory dysfunction. Science 1982; 217:418-488

3. Holman BL, Gibson RE, Hill TC, et al: Muscarinic acetylcholine receptors in Alzheimer's disease. JAMA 1985; 254:3063-3066

4. Holman BL, Hill TC, Polak JF, et al: Cerebral perfusion imaging with iodine-123 labeled amines. Arch Neurol 1984; 41:1060-1066

Tuberculous Peritonitis and Liver Cirrhosis

TO THE EDITOR: I have read with interest the article "Tuberculous Peritonitis Developing in a Case of Documented Peritoneal Carcinomatosis,"[1] which emphasizes the importance of correctly diagnosing tuberculous infections in the presence of concomitant diseases with similar clinical and laboratory features.

Tuberculous peritonitis now has a lower incidence in the more developed countries and often escapes diagnosis.[2] When associated with liver cirrhosis, the diagnostic problems are further increased.[3] I here report two cases associated with liver cirrhosis diagnosed by means of peritoneoscopy, performed under local anesthesia after sedation with diazepam, and peritoneal biopsy.

Reports of Cases

Case 1. The first patient, a 57-year-old woman with cirrhosis, was admitted with ascites resistant to diuretic therapy, intense asthenia and a slight fever. Protein concentration in the ascitic fluid was 1.9 grams per dl; a direct smear was negative for acid-fast bacilli and showed a prevalence of macrophages with some lymphocytes and neutrophil granulocytes. Peritoneoscopy showed medium nodular cirrhosis with numerous signs of endoabdominal portal hypertension. The visceral and parietal peritoneum was congested and covered with small miliary nodules.

Case 2. The second patient, a 44-year-old man with alcoholic cirrhosis, was admitted with ascites resistant to diuretic therapy. A high septic fever and convulsive epileptic crises appeared, and his general condition was serious. Ascitic fluid protein concentration was 3.2 grams per dl and a direct smear showed macrophages, lymphocytes and occasional neutrophil granulocytes; acid-fast bacilli were absent. The peritoneoscopy showed medium nodular hypertrophic cirrhosis

with abundant fibrin in the hepatic surface, and the parietal peritoneum showed scattered small miliary nodules with fibrinous adhesions to the bowel loops. In this case, too, biopsy of these lesions showed caseating granulomas and giant cells. No acid-fast bacilli could be identified.

Certain clinical and laboratory signs found in the two cases studied, such as fever, the exudative character of the ascitic fluid and poor response to diuretic therapy, should hence cause suspicion of an association of a tuberculous peritonitis. This suspicion should not be excluded on the basis of low protein values in the ascitic fluid, as this may be due to dilution by transudation when, as in the first case, there are conspicuous signs of endoabdominal portal hypertension. Peritoneoscopy with biopsy allows a differential diagnosis with other diseases such as peritoneal carcinosis, hepatocarcinoma and nonspecific bacterial peritonitis associated with cirrhosis.[4] In cases of tuberculous peritonitis, peritoneoscopy may constitute an additional risk if there are extensive adhesions between the intestine and the abdominal wall. I believe that the presence of ascitic fluid reduces the risks of this method which allows rapid diagnosis and an early commencement of therapy.

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REFERENCES

1. Markman M: Tuberculous peritonitis developing in a case of documented peritoneal carcinomatosis. West J Med 1985; 143:103-105
2. Hyman S: The enigma of tuberculous peritonitis. Gastroenterology 1962; 42:1-6
3. Geake TM, Spitaels JM, Moshal MG, et al: Peritoneoscopy in the diagnosis of tuberculous peritonitis. Gastrointest Endosc 1981; 27:66-68
4. Buffet C, Pelletier G, Etienne JP: Que rest-t-il des indications de la laparoscopie en 1983? Gastroenterol Clin Biol 1983; 7:134-140

Prostaglandins—Lifesaving Drugs for Postpartum Uterine Atony

TO THE EDITOR: Drs Zipser and Laffi are to be congratulated on their excellent review article "Prostaglandins, Thromboxanes and Leukotrienes in Clinical Medicine" in the October 1985 issue.[1] I found the article to be a timely, superbly written update on the fascinating variety of physiologic actions and potential clinical applications of these substances.

In the section on prostaglandins in reproductive medicine, however, one extremely important application was not mentioned. Prostaglandin (PG) $F_2\alpha$ and its synthetic analog, 15-methylprostaglandin $F_2\alpha$ (carboprost tromethamine [Prostin / 15M]), have an established, lifesaving role in the treatment of postpartum hemorrhage.

Before the availability of these compounds, the only medications for use in the treatment of hemorrhage due to uterine atony were oxytocin and ergot alkaloid derivatives. $PGF_2\alpha$ and carboprost are much more potent uterotonic agents and are successful, in most cases, in controlling atony and hemorrhage when oxytocin or ergots fail.[2-4]

In our experience with carboprost in nearly 100 cases, gastrointestinal side effects—particularly diarrhea—and mild elevation in blood pressure are common. Exacerbation of asthma is a theoretical possibility but one we have not encountered.

$PGF_2\alpha$ is metabolized on first pass through the lungs and thus must be given by direct injection into the uterus (dose: 1 to 5 mg) to be effective. Carboprost is effective when administered by intramuscular or intrauterine injection (dose: 125 to 500 μg). In my view, these are safer and more effective than ergot derivatives and should be used if oxytocin infusion is unsuccessful in controlling bleeding due to uterine atony.

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REFERENCES

1. Zipser RD, Laffi G: Prostaglandins, thromboxanes and leukotrienes in clinical medicine (Medical Progress). West J Med 1985; 143:485-497
2. Toppozoda M, El-Bossaty M, El-Rahman HA, et al: Control of intractable atonic postpartum hemorrhage by 15-methyl prostaglandin F2-alpha. Obstet Gynecol 1981; 58:327-330
3. Hayashi RH, Castillo MS, Noah ML: Management of severe postpartum hemorrhage due to uterine atony using an analogue of prostaglandin F2-alpha. Obstet Gynecol 1981; 58:426-429
4. Catanzarite VA, Moffitt KA, Longmire-Baker M, et al: Acute puerperal uterine inversion—New approaches to management. Obstet Gynecol 1986, in press

Differential Diagnosis of 'Pseudoseptic' Arthritis

TO THE EDITOR: The recent description of five cases of "pseudoseptic" arthritis by Call and colleagues[1] and the ensuing letter by Loge[2] prompted this communication.

Call and co-workers, of the University of Utah School of Medicine, reported five cases of classical rheumatoid arthritis with a superimposed clinical syndrome that mimics septic arthritis but in which microbiological cultures were negative. They concluded that the pathogenesis of "pseudoseptic" arthritis remains unexplained. They considered multiple conditions in seeking an explanation for the cases of "pseudoseptic" arthritis; however, no consideration was given to Lyme disease. Although it would be unusual, it is possible that a patient with classical rheumatoid arthritis could also have Lyme disease. Lyme disease has presented with inflammatory oligoarticular arthritis without preceding rash. Frequently the patient is unaware of a tick bite. Synovial fluid leukocyte counts range up to 100,000 cells per μl with most cells being polymorphonuclear leukocytes.[3] The joint abnormalities may be brief and recurrent[3] and may respond to antibiotics.[4] Culture of the Lyme disease agent, *Borrelia burgdorferi*, requires special techniques.[5]

Two cases of Lyme disease have been reported in Utah.[6] Many cases, however, may go unrecognized in Utah, as is suspected in other states.[6] The tick vector of Lyme disease in California, *Ixodes pacificus*, is known to reside in Utah.[7]

Although it is possible Lyme disease plays a role in the cases presented by Call and associates, it is more the intent of this letter to mention Lyme disease for consideration in the differential diagnosis of septic or "pseudoseptic" arthritis.

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REFERENCES

1. Call RS, Ward JR, Samuelson CO Jr: 'Pseudoseptic' arthritis in patients with rheumatoid arthritis. West J Med 1985 Oct; 143:471-473
2. Loge RV: 'Pseudoseptic' arthritis (Correspondence). West J Med 1986 Jan; 144:87-88